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Comparative Biochemistry and Physiology, Part A 145 (2006) 413–418
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Does overfeeding enhance genotype effects on energy metabolism and lipid deposition in breast muscle of ducks?

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Received 18 October 2005; received in revised form 18 July 2006; accepted 20 July 2006
Available online 13 October 2006

Abstract

We evaluated the effects of genotype (Muscovy, Pekin and their crossbreed hinny and mule ducks) and feeding levels (overfeeding between 12 and 14 weeks of age *vs ad libitum* feeding) on energy metabolism and lipid deposition in breast muscle of ducks. Samples of breast muscle (Pectoralis major) were collected at 14 weeks of age from 8 birds per group. Overfeeding induced an accumulation of lipids in breast muscle (1.5- to 1.7-fold, depending on genotype) mainly induced by triglyceride deposition. It also induced a considerable increase in the amounts (expressed as g/100 g of tissue) of saturated and mono-unsaturated fatty acids (SFA, MUFA), while the amounts of poly-unsaturated fatty acids (PUFA) remained unchanged in hinny and Muscovy ducks or slightly increased in Pekin and mule ducks. In breast muscle, overfeeding decreased the activity of the main enzymes involved in lipogenesis from glucose (glucose-6-phosphate dehydrogenase, G6PDH, malic enzyme, ME, acetyl CoA carboxylase, ACX). Lipoprotein lipase (LPL) activity in Pectoralis major muscle was also significantly decreased (−21%). The ability of muscle tissues to catabolize long-chain fatty acids, as assessed by β-hydroxyacyl CoA dehydrogenase (HAD) activity, was increased in Pectoralis major muscle, as was cytochrome-*c* oxidase (COX) activity. Hybrid and Pekin ducks exhibited higher levels of ACX and LPL activity in Pectoralis major muscle than Muscovy ducks, suggesting a greater ability to synthesise lipids *in situ*, and to take up circulating lipids. Total lipid content in breast muscle of hybrid and Pekin ducks was higher than in that of Muscovy ducks. In hybrid and Pekin ducks, lipid composition of breast muscle was characterized by higher amounts of triglycerides, SFA and MUFA than in Muscovy ducks. Finally, oxidative metabolism was greater in Pectoralis major muscles of hybrid and Pekin ducks than in Muscovy ducks, suggesting an adaptative strategy of muscle energy metabolism according to lipid level.

Keywords: Lipids; Energy metabolism; Muscle; Ducks; Lipoprotein lipase

1. Introduction

In muscle, total lipid level and lipid composition is a balance between the uptake of blood lipids provided by liver lipogenesis or feed, and the muscle metabolism (lipid synthesis and lipid use for energy requirements, Hocquette et al., 1998a). Guy et al. (1999) and Hermier et al. (2003) reported that the different duck genotypes (Pekin, Muscovy and their crossbreed hinny and mule ducks) presented different susceptibility to storing lipids in the muscles during an overfeeding period. Pekin and hybrid ducks exhibited higher lipid content in muscles than Muscovy ducks. *De novo* lipogenesis is essentially hepatic in avian species (Pearce, 1977). As the lipid level in breast meat is two-fold higher in overfed ducks than in lean ducks (Auvergne, 1992), it can be hypothesised that lipogenesis may be enhanced in adipocytes of muscle tissues during overfeeding at different levels according duck genotype. However, in birds the main way to increase lipid level of muscles remains the uptake of blood lipids. Davail et al. (2003a) reported that the activity of

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doi:10.1016/j.cbpa.2006.07.024

plasma lipoprotein lipase (LPL) fell dramatically on the tenth day of the overfeeding period in Muscovy and mule ducks, but remained steady in Pekin ducks. The uptake of plasma lipids into peripheral tissues may therefore be more efficient in Pekin ducks. Finally, lipid content in muscles depends also on the use *in situ* for energy requirements. Zanusso et al. (2003) reported an increased oxidative metabolism in overfed Muscovy ducks compared to *ad libitum*-fed ducks. Therefore we analysed whether this increase occurs in the different duck genotypes after overfeeding.

The aim of this study was to determine the effects of genotypes (Muscovy, Pekin, mule and hinny ducks) and feeding levels (overfeeding *vs ad libitum* feeding) on lipid deposition in breast muscle (quantity and quality) in relationship with muscle energy metabolism (glycolytic and oxidative) and muscle ability in lipid uptake (characterized by lipoprotein lipase activity). A companion paper details hepatic changes in the same birds (Chartrin et al., 2006b).

2. Materials and methods

2.1. Animals and experimental design

Animals and experimental design were described in a companion paper (Chartrin et al., 2006b). At 14 weeks of age, birds were sacrificed. Immediately after bleeding, Pectoralis major muscle was excised and weighed. One sample was quickly frozen in liquid nitrogen and stored at −80 °C until enzyme activity determination. A sample of breast muscle was also frozen and stored at −20 °C to determine lipid level and lipid composition.

2.2. Enzyme activity determination

The activity of malic enzyme (ME, E.C. 1.1.1.40), glucose-6-phosphate dehydrogenase (G6PDH, E.C. 1.1.1.49), acetyl-CoA-carboxylase (ACX, E.C. 6.4.1.2) and cytochrome-*c* oxidase (COX, E.C. 1.9.3.1) were measured in Pectoralis major muscle as previously described in the companion paper (Chartrin et al., 2006b). Lipoprotein lipase activity (LPL, E.C. 3.1.1.34) was determined according to Hocquette et al. (1998b). Activity of three enzymes for glycolytic (lactate dehydrogenase, LDH, E.C. 1.1.2.4) and oxidative (β-hydroxyacyl CoA dehydrogenase, HAD, E.C. 1.1.1.35, and citrate synthase, CS, E.C. 2.3.3.1) energy metabolism were assayed according to Bass et al. (1969).

2.3. Chemical analysis

Total lipids, lipid classes and fatty acid composition were determined in Pectoralis major muscle as previously described in companion paper (Chartrin et al., 2006b).

2.4. Statistical analysis

Data were analysed by analysis of variance using the General Linear Model procedure of SAS (1989). The model included the main effects of genotype, feeding level and their interactions. Significant differences between means of the different groups were shown according to the Newman-Keuls' test. Accepted *p*-value was 0.05. Data for fatty analysis were expressed as percentages. Prior to statistical analysis they were submitted to log transformation.

3. Results

3.1. Breast weight and enzyme activities

By comparison with the other genotypes, Muscovy ducks had the highest muscle weight and Pekin ducks had the lowest one (Table 1). Overfeeding had no effect on muscle growth (Table 1).

By comparison with the other genotypes, Muscovy ducks exhibited the lowest LPL activity and hinny ducks had the

Table 1
Effects of overfeeding and genotype on breast muscle mass (g) and enzyme activity of lipoprotein lipase (LPL, nmol/min/g of muscle), glucose-6-phosphate dehydrogenase (G6PDH, μmol/g of muscle) malic enzyme (ME, μmol/g muscle) and acetyl-CoA-carboxylase (ACX nmol/g muscle) of 14-week-old ducks (mean ±SE, $n=8$)

Genotype	Feeding levels	Breast weight	LPL	G6PDH	ME	ACX
	Overfed	303±33	140±76 b	0.20±0.11	4.27±2.09 b	0.70±0.27 b
	Control	297±29	177±96 a	0.26±0.21	6.18±1.88 a	0.89±0.43 a
Overfeeding effect		ns	*	ns	***	*
Muscovy		403±43 a	128±81 b	0.15±0.05	4.76±1.59	0.60±0.19 b
Hinny		293±27 b	181±86 a	0.21±0.16	4.85±2.43	0.80±0.28a,b
Mule		294±28 b	156±89a,b	0.26±0.20	5.66±2.72	0.89±0.43 a
Pekin		212±26 c	173±94 a	0.30±0.18	5.62±1.92	0.88±0.46 a
Genotype effect		***	*	ns	ns	*
Muscovy	Overfed	408±49	104±65	0.18±0.06	4.35±1.97	0.64±0.21 c
	Control	398±39	151±91	0.13±0.04	5.18±1.06	0.56±0.17 c
Hinny	Overfed	290±25	166±83	0.16±0.14	3.80±2.14	0.81±0.39b,c
	Control	297±29	196±91	0.27±0.17	5.90±2.35	0.79±0.13 c
Mule	Overfed	309±28	130±69	0.19±0.07	4.19±2.56	0.64±0.13 c
	Control	278±30	180±102	0.32±0.27	7.13±2.10	1.14±0.48 a
Pekin	Overfed	208±33	162±79	0.26±0.13	4.73±1.98	0.70±0.28 c
	Control	216±18	183±110	0.33±0.23	6.50±1.49	1.05±0.55a,b
Interaction effect		ns	ns	ns	ns	ns

*, **, ***: significant effect, $P<0.05$, $P<0.01$, $P<0.001$, ns=not significant.
a–c: significant difference between groups for one criterion.

highest values (Table 1). ACX activity was lowest in breast muscle of Muscovy ducks while mule and Pekin ducks had the highest values. Genotype had no significant effect on the activity of G6PDH and ME in breast muscle (Table 1). Muscovy ducks exhibited the highest levels of CS activity and the lowest levels of HAD activity in Pectoralis major muscle (Table 2). Hinny ducks had the lowest level of CS activity. Genotype had no significant effect on LDH activity. The lowest levels of COX activity occurred in Pectoralis major muscle of Pekin ducks.

Overfed ducks had lower LPL, ME, ACX and LDH activity in Pectoralis major than control birds (−21,−22,−31 and −9%, respectively, Tables 1 and 2). Overfeeding significantly increased COX and HAD activity (+18 and +13%, respectively). Overfeeding had no significant effect on G6PDH and CS activity in breast muscle.

3.2. Chemical composition of breast muscle

By comparison with other genotypes, Muscovy ducks exhibited the lowest lipid, triglyceride and phospholipid levels in breast muscle and Pekin ducks the highest levels of lipids and triglycerides (Table 3). The cholesterol levels were similar for all genotypes.

Overfeeding induced a significant increase in lipid and triglyceride levels of Pectoralis major (1.6 and 2.0-fold, respectively, Table 3).

Table 2
Effects of overfeeding and genotype on the enzymatic activity (IU/g muscle) of lactate dehydrogenase (LDH), citrate synthase (CS), β-hydroyacyl-CoA dehydrogenase (HAD) and cytochrome-*c* oxidase (COX) in Pectoralis major muscles of 14-week-old ducks (mean±SE, $n=8$)

Genotype	Feeding levels	HAD[a]	LDH[a]	CS[a]	COX[b]
	Overfed	16.58±2.90 a	439±63 b	8.85±1.10	31.51±12.07 a
	Control	14.67±2.69 b	484±55 a	8.92±2.20	26.66±8.32 b
Overfeeding effect		*	**	ns	*
Muscovy		11.64±3.02 b	497±73	10.16±1.79a	30.16±8.72 a
Hinny		16.80±2.11 a	447±41	7.82±1.49 b	31.21±9.93 a
Mule		16.74±2.48 a	448±72	8.87±1.84a,b	31.77±12.48 a
Pekin		17.32±3.56 a	452±49	8.68±1.93a,b	23.21±9.25 b
Genotype effect		***	ns	**	*
Muscovy	Overfed	13.70±3.15	506±91 a	9.91±1.18	33.04±9.79
	Control	9.58±3.09	489±56a,b	10.41±2.33	27.28±6.80
Hinny	Overfed	17.20±1.80	397±37 c	7.62±1.18	30.64±12.79
	Control	16.39±2.51	498±47a,b	8.03±1.84	31.78±6.64
Mule	Overfed	17.64±2.39	441±68 a,b,c	9.28±1.23	37.71±12.54
	Control	15.84±2.73	456±82 a,b,c	8.46±2.39	25.84±9.65
Pekin	Overfed	17.77±4.27	410±60b,c	8.58±1.04	24.67±10.80
	Control	16.86±2.97	495±38a,b	8.79±2.63	21.75±7.69
Interaction effect		ns	*	ns	ns

*, **, ***: significant effect, $P<0.05$, $P<0.01$, $P<0.001$, ns=not significant.
a–c: significant difference between groups for one criterion.
[a] Expressed as mmol NADH/min/g tissue.
[b] Expressed as μmol cytochrome-*c* oxidase/min/g tissue.

Table 3
Effects of overfeeding and genotype on total lipid, triglyceride, phospholipid and cholesterol levels (g/100 g tissue) in breast muscles of 14-week-old ducks sacrificed more than 12 h after their last meal (mean±SE, $n=8$)

Genotype	Feeding levels	Lipids	Triglycerides	Phospholipids	Cholesterol
	Overfed	5.59±0.81 a	4.08±0.73 a	1.37±0.17	0.14±0.04
	Control	3.46±0.82 b	2.02±0.53 b	1.30±0.28	0.14±0.05
Overfeeding effect		***	***	ns	ns
Muscovy		2.95±0.42 d	1.69±0.36 d	1.14±0.14 b	0.12±0.04
Hinny		4.90±1.19 b	3.27±0.79 b	1.48±0.39 a	0.15±0.06
Mule		4.18±0.75 c	2.73±0.66 c	1.32±0.18 a	0.14±0.04
Pekin		6.08±0.74 a	4.52±0.71 a	1.40±0.14 a	0.16±0.03
Genotype effect		***	***	**	ns
Muscovy	Overfed	3.65±0.51	2.37±0.47 d	1.15±0.17	0.13±0.05
	Control	2.26±0.36	1.02±0.24 e	1.13±0.10	0.12±0.04
Hinny	Overfed	5.92±0.98	4.35±0.76 b	1.43±0.26	0.14±0.05
	Control	3.87±1.44	2.19±0.87 d	1.53±0.51	0.16±0.08
Mule	Overfed	5.24±0.96	3.66±0.91 c	1.42±0.11	0.16±0.04
	Control	3.13±0.54	1.79±0.32 d	1.21±0.24	0.13±0.03
Pekin	Overfed	7.57±0.85	5.95±0.86 a	1.46±0.13	0.15±0.02
	Control	4.59±0.68	3.09±0.58 c	1.33±0.15	0.17±0.03
Interaction effect		ns	*	ns	ns

*, **, ***: significant effect, $P<0.05$, $P<0.01$, $P<0.001$, ns=not significant.
a–e: significant difference between groups for one criterion.

The main fatty acids in breast muscle was C16:0 (23 to 26%) and C18:0 (7 to 10%) among saturated fatty acids (SFA), C18:1 n-9 (30 to 46%) and C16:1 n-7 (2 to 4%) among mono-unsaturated fatty acids (MUFA), C18:2 n-6 (11 to 16%) and C20:4 n-6 (3 to 7%) among poly-unsaturated fatty acids (PUFA, Table 4). Breast muscle contained high proportions of n-6 fatty acids and very low proportions of n-3 fatty acids. Pekin ducks exhibited the highest proportions of MUFA and the lowest proportions of SFA and PUFA (Table 4). Muscovy ducks exhibited the highest proportions of SFA and PUFA and the lowest proportions of MUFA. Calculating amounts of fatty acids per 100 g of tissue showed that Muscovy ducks had the lowest levels of all classes of fatty acids and the Pekin ducks the highest levels (Fig. 1).

Overfeeding induced an increase in proportions of MUFA and a decrease in proportions of PUFA (Table 4). Calculating amounts of fatty acids per 100 g of tissue showed that overfeeding finally induced large increases in SFA and MUFA levels particularly in C16:0, C:18:0, C16:1 and C18:1 fatty acids (Fig. 1). PUFA levels remained practically unchanged in Muscovy and hinny ducks and increased in mule and Pekin ducks, particularly in C18:2 fatty acid.

4. Discussion

4.1. Lipogenesis and lipid deposition in breast muscle

Whatever the feeding level, total lipid content of breast muscle was always lower in Muscovy ducks than in the other genotypes. This data was in agreement with the observations of Hermier et al. (2003) and Chartrin et al. (2006a). Breast muscle of Muscovy ducks contained also lower amounts of

Table 4
Effects of overfeeding and genotype on the fatty acid composition (% total fatty acids) of total lipids in breast muscles of 14-week-old ducks sacrificed more than 12 h after their last meal (mean±SE, $n=8$)

Fatty acids	Muscovy		Hinny		Mule		Pekin		Genotype	Overfeeding
	Overfed	Control	Overfed	Control	Overfed	Control	Overfed	Control	effect	effect
C13: 0	0.22±0.30 b	1.48±1.00 a	0.05±0.15 b	0.32±0.35 b	0.16±0.23 b	0.47±0.56 b	0.11±0.19 b	0.40±0.27 b	***	***
C14: 0	0.49±0.36	0.43±0.66	0.37±0.15	0.14±0.25	0.44±0.05	0.41±0.62	0.35±0.15	0.46±0.34	ns	ns
C16: 0	26.25±1.08	24.19±2.37	25.66±1.61	24.40±2.34	26.08±1.07	24.97±1.94	23.24±0.84	23.40±1.08	**	*
C17: 0	nd	0.12±0.35	nd	nd	nd	0.13±0.37	nd	nd	ns	ns
C18: 0	10.06±0.84 a,b	11.21±1.41 a	9.75±0.71 b	9.40±1.44 b	10.39±0.94 a,b	9.71±1.16 b	9.22±0.64 b	7.46±0.78 c	***	ns
C20: 0	nd	0.17±0.34	nd	nd	nd	0.26±0.57	nd	nd	ns	ns
C24: 0	0.95±0.11	1.60±0.88	0.57±0.08	1.09±0.17	0.83±0.28	0.96±0.81	0.49±0.04	0.72±0.30	**	**
Σ SFA	37.97±1.22	39.20±1.59	36.40±1.80	35.35±3.31	37.91±1.19	36.92±1.82	33.39±1.31	32.42±1.15	***	ns
C15: 1 n-5	1.29±0.65 c	3.21±0.60 a	0.86±0.19 c,d	1.82±0.41 b	1.24±0.28 c	2.11±0.64 b	0.60±0.12 d	1.09±0.28 c,d	***	***
C16: 1 n-7	3.10±0.24 b	2.47±0.76 c	3.20±0.25 b	3.24±0.49 b	3.25±0.33 b	3.29±0.88 b	3.60±0.36 b	4.18±0.61 a	***	ns
C17: 1 n-7	0.53±0.12	0.62±0.57	0.44±0.05	0.42±0.47	0.56±0.08	0.38±0.43	0.40±0.04	0.40±0.25	ns	ns
C18: 1 n-9	41.37±2.03 b	30.58±2.07 d	43.89±2.66 b	37.38±3.79 c	40.60±1.02 b	36.06±3.10 c	46.58±2.10 a	43.51±2.17 b	***	***
C20: 1 n-9	0.06±0.18	0.31±0.60	0.13±0.24	nd	nd	0.37±0.70	0.44±0.19	0.08±0.22	ns	ns
Σ MUFA	46.35±1.61 c	37.20±2.24 e	48.52±2.61 b,c	42.86±3.42 d	45.66±0.92 c	42.22±3.18 d	51.62±1.79 a	49.26±2.25a,b	***	***
C18: 2 n-6	11.62±0.82 d	16.04±1.02 a	11.26±0.50 d	15.47±0.95 a,b	11.71±0.78 d	14.59±1.43 b	11.30±0.70 d	13.49±0.83 c	***	***
C18: 3 n-3	0.08±0.16	0.30±0.65	0.33±0.14	0.31±0.34	0.20±0.21	0.33±0.72	0.46±0.04	0.64±0.35	ns	ns
C20: 3 n-6	0.29±0.34	nd	0.33±0.23	nd	0.34±0.24	0.19±0.54	0.55±0.17	0.05±0.13	ns	*
C20: 4 n-6	3.70±0.60 c,d	7.26±1.42 a	3.15±0.50 c,d	6.02±0.98 b	4.18±0.61 c	5.75±1.75 b	2.68±0.37 d	4.14±1.11 c	***	***
Σ PUFA	15.69±1.39 d	23.60±1.86 a	15.08±1.00 d	21.80±1.26 b	16.43±1.32 d	20.86±3.27 b	14.99±1.03 d	18.32±1.77 c	***	***
UFA/SFA	1.64±0.09	1.55±0.10	1.75±0.15	1.85±0.29	1.64±0.08	1.71±0.12	2.00±0.12	2.09±0.11	***	ns
PUFA/SFA	0.41±0.04	0.60±0.06	0.41±0.02	0.62±0.07	0.44±0.05	0.57±0.10	0.45±0.03	0.56±0.05	ns	***
Σ n-6	15.60±1.45 c,d	23.30±1.95 a	14.75±0.97 d	21.49±1.40 b	16.23±1.41 c,d	20.53±3.06 b	14.53±1.00 d	17.68±1.82 c	***	***
Σ n-3	0.08±0.16	0.30±0.65	0.33±0.14	0.31±0.34	0.20±0.21	0.33±0.72	0.46±0.04	0.64±0.35	ns	ns

*, **, ***: significant effect, $P<0.05$, $P<0.01$, $P<0.001$, ns=not significant, nd=not detected.
a–e: significant difference between groups for one criterion, $P<0.05$ (interaction between genotype and overfeeding) SFA, MUFA, PUFA=Saturated, Mono-Unsaturated and Poly-Unsaturated Fatty Acids.

phospholipids than the other genotypes. These lipids are mainly located in membranes including the sarcolemma. Chartrin et al. (2005) reported that Muscovy ducks had muscle fibres exhibiting larger cross-sectional area than the other genotypes. Within one given amount of muscle, Muscovy duck will therefore have a lower number of muscle fibres and by a way of consequence a lower membrane perimeter to fibre volume ratio and phospholipid content than the other genotypes. The difference between the extreme genotypes, Muscovy and Pekin ducks, in terms of lipid content in breast muscle was not reinforced by overfeeding. The increase in total lipid content induced by genotype or by overfeeding mainly resulted from a deposition of triglycerides and MUFA. Oleic and palmitoleic acids are the main fatty acids synthesised per birds (Klasing, 1998). SFA also increased, mainly palmitic and stearic acids provided per feed. In spite of large amounts of linoleic acid representing 57% of total fatty acids provided by the overfeeding diet (Chartrin et al., 2006a), PUFA amounts in breast muscle slightly increased. Lipid composition in muscle was therefore mainly influenced by lipogenesis than feed composition. Moreover, lipogenesis, estimated by G6PDH, ME and ACX activity, was very low in Pectoralis major muscle when comparing with the activity values of these enzymes in the livers of ducks (Chartrin et al., 2006b) or geese (Mourot et al., 2000). Muscovy duck had lower ACX activity in breast muscle than the other genotypes, suggesting a lower lipogenesis muscle activity which could partly explain its lower lipid content. The hybrid and Pekin ducks had similar ACX activity. These observations are in agreement with the greater surface occupied by adipocytes in cross-sections of breast muscle from Pekin and hybrid ducks reported by Chartrin et al. (2005). G6PDH, ME and ACX activity in overfed ducks was lower than in *ad libitum*-fed ducks. The lipogenesis activity in breast muscle seemed therefore inhibited during overfeeding by the accumulation of lipids reinforcing the role of hepatic lipogenesis and the muscle ability in the uptake of circulating lipids to modulate lipid content and lipid composition in muscle.

4.2. Regulation of LPL activity

Surprisingly, muscle LPL activity was lower in all overfed ducks than in *ad libitum*-fed birds. It is generally accepted that overfeeding leading to obesity in mammals increases LPL activity (Granneman and Wade, 1983; Dugail et al., 1986; Cleary and Phillips, 1996). However, this up-regulation of LPL activity depends on the tissue (adipose or muscle) and the origin of the obesity, as LPL activity is under the control of different factors such as insulin and leptin (Picard et al., 1998, 2002). For example, LPL activity in white inguinal and epididymal and brown interscapular adipose tissues of control obese mice (ob/ob) was at least 2-fold greater than in lean mice, but at similar levels in the Vastus lateralis muscle (Picard et al., 1998). Davail et al. (2003a) measured plasma LPL activity in Pekin, mule and Muscovy ducks on the 1st and 10th day of overfeeding, 90 min after a meal. They reported that levels of LPL activity were similar in the three genotypes at the beginning of the

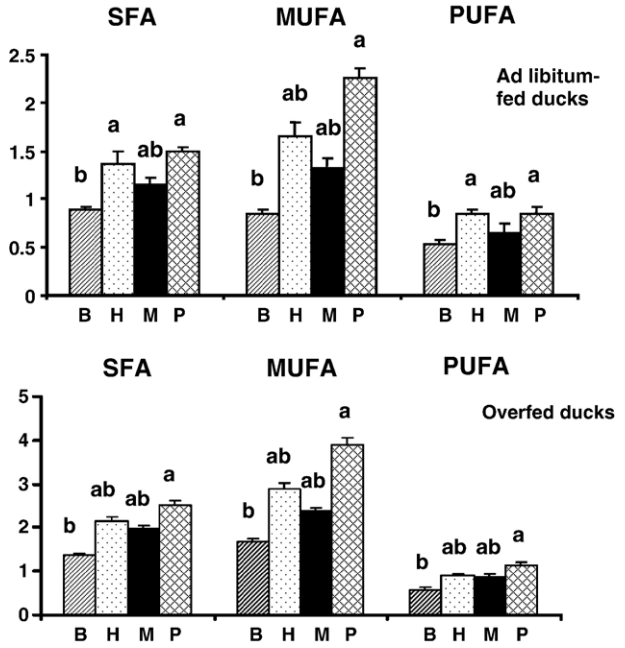

Fig. 1. Effects of genotype on the amounts of saturated, mono-unsaturated and poly-unsaturated fatty acids (SFA, MUFA, PUFA, g/100 g tissue) in breast muscles of 14-week-old ducks (Muscovy=B, Hinny=H, Mule=M, Pekin=P) fed ad libitum or overfed.

overfeeding period (3.2 to 4.5 μmol of palmitate/h/ml) then strongly decreased in Muscovy and mule ducks (to 14 and 28% of the initial values, respectively) but remained high for Pekin ducks (79% of the initial value) after 10 days of overfeeding. In Pekin ducks the insulin/glucagon ratio was 5-fold increased after 10 days of overfeeding whereas it remained steady in Muscovy and mule ducks, suggesting that insulin stimulation of LPL activity only occurred in Pekin ducks. Comparing different levels of overfeeding (moderate *vs* intensive) in mule and Pekin ducks, Davail et al. (2003b) reported that plasma LPL activity decreased between the 1st and 10th day of overfeeding except in Pekin ducks submitted to intensive overfeeding. In our study, we also found lower levels of LPL activity in Pectoralis major muscle of Muscovy ducks, confirming the previous observations and partly explaining the lower lipid deposition in muscle tissues for this species. LPL activity of hybrid ducks was similar to that of Pekin ducks. By considering GK, G6PDH, ME and ACX activity in liver, plasma lipid level (Chartrin et al., 2006b), LPL activity and lipid content in breast muscle (present data), hinny ducks had closer characteristics to those of Pekin ducks than mule ducks. Nevertheless, regulation of LPL activity in the different genotypes during overfeeding needs further investigation. It will be interesting to study the kinetics of LPL activity in response to a meal as well as its evolution throughout the overfeeding period.

4.3. Regulation of energy metabolism

Overall oxidative ability, estimated by COX activity, was increased in Pectoralis major muscle following overfeeding. The muscle tissues adapted their energy metabolism, increasing their oxidative metabolism (characterized by CS or HAD activity, depending on the muscle type), and decreasing their glycolytic metabolism (characterized by a lower LDH activity). Zanusso et al. (2003) also observed an increase in HAD and CS activity in breast muscle from overfed compared to *ad libitum*-fed Muscovy ducks. Our general interpretation is that muscle tissues adapt their metabolism to use more fat as the energy source following overfeeding due to greater fat availability (as demonstrated by higher lipid levels in the plasma, Chartrin et al., 2006b) and reduce utilisation of glucose. HAD activity was lower in muscles of Muscovy ducks, which also exhibited lower lipid levels than the other genotypes. This is line with the general idea that skeletal muscles rich in fat are also more oxidative (Hocquette et al., 1998a).

From this study and the data reported by Chartrin et al. (2006b), we can conclude that lipid content and lipid composition in duck muscle depend mainly from muscle ability in the uptake of circulating lipids than muscle lipogenesis activity. Levels of muscle lipoprotein lipase activity were lower in the Muscovy duck than in the other genotypes, particularly at the end of the overfeeding period, which might enhance the return of circulating triglycerides to the liver. These findings could partly explain the greater susceptibility of the Muscovy duck to hepatic steatosis while the Pekin duck exhibits greater fattiness in adipose and muscle tissues (Chartrin et al., 2006a,b). Davail et al. (2000) demonstrated in overfed Landes geese that fatty liver weight was negatively correlated with plasma LPL activity, suggesting that lipoprotein triacylglycerols not hydrolyzed by LPL may return to the liver and contribute to hepatic steatosis. In hinny and mule ducks the high level of LPL activity enables them to deposit high level of lipids in breast muscle. Lipogenesis activity was also higher in breast muscle of hybrid and Pekin ducks. Faced with increases in lipid levels, muscles adapted their energy metabolism by increasing their oxidative capacity. During overfeeding lipogenesis mainly occurred in the liver (Chartrin et al., 2006b) and the low activity detected in muscle tissue was further decreased.

Acknowledgements

We thank Grimaud and particularly Bernard Retailleau and Magali Banchet for producing and providing the animals. We also thank the technical staff of INRA Artiguères for rearing and overfeeding ducks and Thierry Bordeau for his expert technical assistance in animal dissection and sample preparation.

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